CLAIMS

1. A method for proliferating cardiomyocytes comprising a step of introducing

(a) cyclin,

(b) cyclin-dependent kinase, and

(c) one or a plurality selected from the group consisting of a gene encoding a factor that inhibits the production, function or action of Cip/Kip family protein, and a nucleic acid that inhibits the production of Cip/Kip family protein, into cardiomyocytes, and

a step of subsequently culturing or maintaining said cells.

2. A method for proliferating cardiomyocytes comprising a step of introducing

(a) cyclin,

(b) cyclin-dependent kinase, and

(c) one or a plurality selected from the group consisting of a gene encoding a factor that inhibits the production, function or action of Cip/Kip family protein, and a nucleic acid that inhibits the production of Cip/Kip family protein, into cardiomyocytes *in vitro*, and

a step of subsequently culturing said cells.

3. A method for proliferating cardiomyocytes comprising a step of introducing

(a) cyclin,

(b) cyclin-dependent kinase, and

(c) one or a plurality selected from the group consisting of a gene encoding a factor that inhibits the production, function or action of Cip/Kip family protein, and a nucleic acid that inhibits the production of Cip/Kip family protein, into cardiomyocytes *in vivo*, and

a step of subsequently maintaining said cells.

4. The method of any one of claims 1 to 3, wherein said cyclin is a cyclin capable of activating CDK4 or CDK6 of mammals.

5. The method of claim 4, wherein said cyclin is cyclin D of mammals.

6. The method of any one of claims 1 to 5, wherein said cyclin-dependent kinase is a cyclin-dependent kinase to be activated by cyclin D.

7. The method of claim 6, wherein said cyclin dependent kinase is CDK4 or CDK6.

8. The method of any one of claims 1 to 7, wherein the Cip/Kip

family protein is $p27^{Kip1}$.

9. The method of any one of claims 1 to 8, wherein the factor that inhibits the production, function, or action of Cip/Kip family protein is a factor with an action to promotes the degradation of the Cip/Kip family protein.

10. The method of claim 9, wherein the factor with an action to promote the degradation of the Cip/Kip family protein is a component of ubiquitin ligase.

11. The method of claim 10, wherein the component of ubiquitin ligase is an F-box factor capable of binding to the Cip/Kip family protein.

12. The method of claim 11, wherein the F-box factor capable of binding to the Cip/Kip family protein is Skp2.

13. The method of any one of claims 1 to 12, wherein the nucleic acid that inhibits the production of Cip/Kip family protein is siRNA specific to a gene encoding the Cip/Kip family protein.

14. The method of claim 13, wherein the nucleic acid that inhibits the production of Cip/Kip family protein is siRNA

specific to the $p27^{Kip1}$ gene.

15. The method of any one of claims 1 to 14, comprising introducing the genes into cardiomyocytes, using a viral vector or liposome.

16. The method of any one of claims 1 to 15, wherein at least one of the cyclin gene and cyclin-dependent kinase gene is tagged with a nucleotide sequence encoding a nuclear localization signal.

17. A vector comprising

(a) a cyclin gene

(b) a cyclin-dependent kinase gene, and

(c) one or a plurality selected from the group consisting of a gene encoding a factor that inhibits the production, function, or action of Cip/Kip family protein, and a nucleic acid sequence that inhibits the production of Cip/Kip family protein.

18. The vector of claim 17, wherein the cyclin is a cyclin capable of activating CDK4 or CDK6 of mammals.

19. The vector of claim 18, wherein the cyclin is cyclin D of mammals.

20. The vector of any one of claims 17 to 19, wherein the cyclin-dependent kinase is a cyclin-dependent kinase to be activated by cyclin D.

21. The vector of claim 20, wherein the cyclin-dependent kinase is CDK4 or CDK6.

22. The vector of any one of claims 17 to 21, wherein the factor that inhibits the production, function, or action of Cip/Kip family protein is a factor with an action to promote the degradation of the Cip/Kip family protein.

23. The vector of claim 22, wherein the factor with an action to promote the degradation of the Cip/Kip family protein is a component of ubiquitin ligase.

24. The vector of claim 23, wherein the component of ubiquitin ligase is an F-box factor capable of binding to the Cip/Kip family protein.

25. The vector of claim 24, wherein the F-box factor capable of binding to the Cip/Kip family protein is Skp2.

26. The vector of any one of claims 17 to 25, wherein the

nucleic acid that inhibits the production of Cip/Kip family protein is siRNA specific to a gene encoding the Cip/Kip family protein.

27. The vector of claim 26, wherein the nucleic acid that inhibits the production of Cip/Kip family protein is siRNA that is specific to $p27^{Kip1}$ gene.

28. The vector of any one of claims 17 to 27, wherein at least one of the cyclin gene and cyclin-dependent kinase gene is tagged with a nucleotide sequence encoding a nuclear localization signal.

29. A pharmaceutical composition for use in a treatment of cardiac disorder comprising the vector of any one of claims 17 to 28.

30. The pharmaceutical composition of claims 29, wherein the cardiac disorder is myocardial infarction, ischemic heart disease, congestive heart failure, hypertrophic cardiomyopathy, dilated cardiomyopathy, myocarditis, or chronic heart failure.

31. Cardiomyocyte obtained by the method of any one of claims 1 to 16.

32. A method of treating a cardiac disorder comprising injecting the pharmaceutical composition of claim 29, or transplanting the cardiomyocytes of claim 31 into a site of disorder of a subject having a cardiac disorder, and retaining and proliferating the cardiomyocytes at said site.

33. The method of claim 32, wherein the cardiac disorder is myocardial infarction, ischemic heart disease, congestive heart failure, hypertrophic cardiomyopathy, dilated cardiomyopathy, myocarditis, or chronic heart failure.